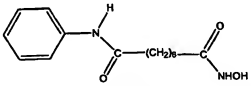
What is claimed is:

1. A method of treating cancer in a subject in need thereof, comprising the step of administering to the subject a first amount of suberoylanilide hydroxamic acid (SAHA) or a pharmaceutically acceptable salt or hydrate thereof, represented by the structure:



in a first treatment procedure, and a second amount of an anti-cancer agent in a second treatment procedure, wherein the first and second amounts together comprise a therapeutically effective amount.

2. The method of claim 1, wherein said anti-cancer agent is a histone deacetylase (HDAC) inhibitor, an alkylating agent, an antibiotic agent, an antimetabolic agent, a hormonal agent, a plant-derived agent, an anti-angiogenic agent, a differentiation inducing agent, a cell growth arrest inducing agent, an apoptosis inducing agent, a cytotoxic agent, a biologic agent, a gene therapy agent, or any combination thereof.

3. The method of claim 1, wherein said anti-cancer compound is a histone deacetylase (HDAC) inhibitor.

4. The method of claim 3, wherein said HDAC inhibitor is a hydroxamic acid derivative, a Short Chain Fatty Acid (SCFA), a cyclic tetrapeptide, a benzamide derivative, or an electrophilic ketone derivative.

5. The method of claim 3, wherein said HDAC inhibitor is a hydroxamic acid derivative selected from the group consisting of SAHA, Pyroxamide, CBHA, Trichostatin A (TSA), Trichostatin C, Salicylbishydroxamic Acid, Azelaic Bishydroxamic Acid (ABHA), Azelaic-1-Hydroxamate-9-Anilide (AAHA), 6-(3-Chlorophenylureido) carpoic Hydroxamic Acid (3Cl-UCHA), Oxamflatin, A-161906, Scriptaid, PXD-101, LAQ-824, CHAP, MW2796, and MW2996.

6. The method of claim 3, wherein said HDAC inhibitor is a Cyclic Tetrapeptide selected from the group consisting of Trapoxin A, FR901228 (FK 228 or Depsipeptide), FR225497, Apicidin, CHAP, HC-Toxin, WF27082, and Chlamydocin.

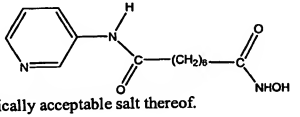
7. The method of claim 3, wherein said HDAC inhibitor is a Short Chain Fatty Acid (SCFA) selected from the group consisting of Sodium Butyrate, Isovalerate, Valerate, 4 Phenylbutyrate (4-PBA), Phenylbutyrate (PB), Propionate, Butyramide, Isobutyramide, Phenylacetate, 3-Bromopropionate, Tributyrin, Valproic Acid and Valproate.

8. The method of claim 3, wherein said HDAC inhibitor is a Benzamide derivative selected from the group consisting of CI-994, MS-27-275 (MS-275) and a 3'-amino derivative of MS-27-275.

9. The method according to claim 3, wherein said HDAC inhibitor is an electrophilic ketone derivative selected from the group consisting of a trifluoromethyl ketone and an α-keto amide.

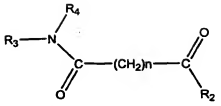
10. The method according to claim 3, wherein said HDAC inhibitor is a natural product, a psammaplin or Depudecin.

11. The method according to claim 3, wherein said HDAC inhibitor is pyroxamide, represented by the structure:



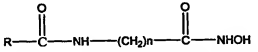
or a pharmaceutically acceptable salt thereof.

12. The method of claim 3, wherein said HDAC inhibitor is represented by the structure:



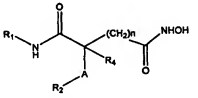
wherein R_3 and R_4 are independently a substituted or unsubstituted, branched or unbranched alkyl, alkenyl, cycloalkyl, aryl, alkyloxy, aryloxy, arylalkyloxy, or pyridine group, cycloalkyl, aryl, aryloxy, arylalkyloxy, or pyridine group, or R_3 and R_4 bond together to form a piperidine group; R_2 is a hydroxylamino group; and n is an integer from 5 to 8.

13. The method of claim 3, wherein said HDAC inhibitor is represented by the structure:



wherein R is a substituted or unsubstituted phenyl, piperidine, thiazole, 2-pyridine, 3- pyridine or 4-pyridine and n is an integer from 4 to 8.

14. The method of claim 3, wherein said HDAC inhibitor is represented by the structure:



wherein A is an amide moiety, R_1 and R_2 are each selected from substituted or unsubstituted aryl, arylalkyl, naphthyl, pyridineamino, 9-purine-6-amino, thiazoleamino, aryloxy, arylalkyloxy, pyridyl, quinolinyl or isoquinolinyl; R_4 is hydrogen, a halogen, a phenyl or a cycloalkyl moiety and n is an integer from 3 to 10.

15. The method of claim 1, wherein the anti-cancer agent is an alkylating agent selected from the group consisting of bischloroethylamines, aziridines, alkyl alkone sulfonates, nitrosoureas, nonclassic alkylating agents and platinum compounds.

16. The method of claim 1, wherein the anti-cancer agent is an antibiotic agent selected from the group consisting of doxorubicin, daunorubicin, epirubicin, idarubicin and anthracenedione, mitomycin C, bleomycin, dactinomycin, and plicatomycin.

17. The method of claim 1, wherein the anti-cancer agent is an antimetabolic agent selected from the group consisting of floxuridine, fluorouracil, methotrexate, leucovorin, hydroxyurea, thioguanine, mercaptopurine, cytarabine, pentostatin, fludarabine phosphate, cladribine, asparaginase, and gemcitabine.

18. The method of claim 17, wherein said antimetabolic agent is gemcitabine.

19. The method of claim 1, wherein the anti-cancer agent is an hormonal agent selected from the group consisting of an estrogen, a progestogen, an antiesterogen, an androgen, an antiandrogen, an LHRH analogue, an aromatase inhibitor, diethylstibestrol, tamoxifen, toremifene, fluoxymesterol, raloxifene, bicalutamide, nilutamide, flutamide, aminoglutethimide, tetrazole, ketoconazole, goserelin acetate, leuprolide, megestrol acetate, and mifepristone.

20. The method of claim 1, wherein the anti-cancer agent is a plant-derived agent selected from the group consisting of vincristine, vinblastine, vindesine, vinzolidine, vinorelbine, etoposide teniposide, paclitaxel and docetaxel.

21. The method of claim 1, wherein the anti-cancer agent is a biologic agent is selected from the group consisting of immuno-modulating proteins, monoclonal antibodies against tumor antigens, tumor suppressor genes, and cancer vaccines.

22. The method of claim 21, wherein the immuno-modulating protein is selected from the group consisting of interleukin 2, interleukin 4, interleukin 12, interferon El interferon D, interferon alpha, erythropoietin, granulocyte-CSF, granulocyte, macrophage-CSF, bacillus Cahnette-Guerin, levamisole, and octreotide.

23. The method of claim 21, wherein the tumor suppressor gene is selected from the group consisting of DPC-4, NF-1, NF-2, RB, p53, WTl, BRCA, and BRCA2.

24. The method of claim 1, wherein the anti-cancer agent is a differentiation inducing agent.

25. The method of claim 1, wherein the therapeutic effect of SAHA and said anti-cancer agent is additive.

26. The method of claim 1, wherein SAHA sensitizes cancer cells in the patient to said anti-cancer agent.

27. The method of claim 1, wherein said anti-cancer agent sensitizes cancer cells in the patient to SAHA.

28. The method of claim 1, wherein SAHA and said anti-cancer agent are administered simultaneously.

29. The method of claim 1, wherein SAHA and said anti-cancer agent are administered sequentially.

30. The method of claim 29, wherein SAHA is administered prior to administering said anti-cancer agent.

31. The method of claim 29, wherein SAHA is administered after administering said anti-cancer agent.

32. The method of claim 1, wherein SAHA is administered orally, parenterally, intraperitoneally, intravenously, intraarterially, transdermally, sublingually, intramuscularly, rectally, transbuccally, intranasally, liposomally, via inhalation, vaginally, intraoccularly, via local delivery by catheter or stent, subcutaneously, intraadiposally, intraarticularly, intrathecally, or in a slow release dosage form.

33. The method of claim 1, wherein said anti-cancer agent is administered orally, parenterally, intraperitoneally, intravenously, intraarterially, transdermally,

sublingually, intramuscularly, rectally, transbuccally, intranasally, liposomally, via inhalation, vaginally, intraoccularly, via local delivery by catheter or stent, subcutaneously, intraadiposally, intraarticularly, intrathecally, or in a slow release dosage form.

34. The method of claim 1, wherein SAHA is administered orally in a pharmaceutical composition comprising SAHA and a pharmaceutically acceptable carrier or diluent.

35. The method of claim 34, wherein said SAHA composition is contained within a gelatin capsule.

36. The method of claim 35, wherein said carrier or diluent is microcrystalline cellulose.

37. The method of claim 36, wherein said SAHA composition further comprises sodium croscarmellose as a disintegrating agent.

38. The method of claim 37, wherein said SAHA composition further comprises magnesium stearate as a lubricant.

39. The method of claim 34, wherein said SAHA composition is administered to the subject at a total daily dosage of between about 25-4000 mg/m^2.

40. The method of claim 34, wherein said SAHA composition is administered once-daily, twice-daily or three times-daily.

41. The method of claim 40, wherein said SAHA composition is administered once daily at a dose of about 200-600 mg.

42. The method of claim 40, wherein said SAHA composition is administered twice daily at a dose of about 200-400 mg.

43. The method of claim 40, wherein said SAHA composition is administered three times daily at a dose of about 200-400 mg.

44. The method of claim 40, wherein said SAHA composition is administered twice daily at a dose of about 200-400 mg intermittently.

45. The method of claim 44, wherein said SAHA composition is administered three to five days per week.

46. The method of claim 44, wherein said SAHA composition is administered three days a week.

47. The method of claim 46, wherein said SAHA composition is administered at a dose of about 200 mg.

48. The method of claim 46, wherein said SAHA composition is administered at a dose of about 300 mg.

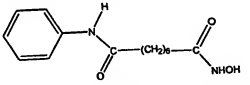
49. The method of claim 46, wherein said SAHA composition is administered at a dose of about 400 mg.

50. The method of claim 1, wherein the cancer is selected from the group consisting of a leukemia, a lymphoma, a myeloma, a sarcoma, a carcinoma, a solid tumor or any combination thereof.

51. The method of claim 1, wherein the cancer is selected from the group consisting of cutaneous T-cell lymphoma (CTCL), noncutaneous peripheral T-cell lymphoma, lymphoma associated with human T-cell lymphotrophic virus (HTLV), adult T-cell leukemia/lymphoma (ATLL), acute lymphocytic leukemia, acute nonlymphocytic leukemia, chronic lymphocytic leukemia, chronic myelogenous leukemia, Hodgkin's disease, non-Hodgkin's lymphoma, multiple myeloma, mesothelioma, childhood solid tumors such as brain neuroblastoma, retinoblastoma, Wilms' tumor, bone cancer and soft-tissue sarcomas, common solid tumors of adults such as head

and neck cancers (e.g., oral, laryngeal and esophageal), genito urinary cancers (e.g., prostate, bladder, renal, uterine, ovarian, testicular, rectal and colon), lung cancer, breast cancer, pancreatic cancer, melanoma and other skin cancers, stomach cancer, brain cancer, liver cancer, adrenal cancer, kidney cancer, thyroid cancer, basal cell carcinoma, squamous cell carcinoma of both ulcerating and papillary type, metastatic skin carcinoma, medullary carcinoma, osteo sarcoma, Ewing's sarcoma, veticulum cell sarcoma, Kaposi's sarcoma, neuroblastoma and retinoblastoma.

52. A method of treating cancer in a subject in need thereof, comprising the step of administering to the subject a first amount comprising a total daily dose of up to about 800 mg suberoylanilide hydroxamic acid (SAHA) or a pharmaceutically acceptable salt or hydrate thereof, represented by the structure:



in a first treatment procedure, and a second amount of an anti-cancer agent in a second treatment procedure, wherein the first and second amounts together comprise a therapeutically effective amount.

53. The method of claim 52, wherein said anti-cancer agent is a histone deacetylase (HDAC) inhibitor, an alkylating agent, an antibiotic agent, an antimetabolic agent, a hormonal agent, a plant-derived agent, an anti-angiogenic agent, a differentiation inducing agent, a cell growth arrest inducing agent, an apoptosis inducing agent, a cytotoxic agent, a biologic agent, a gene therapy agent, or any combination thereof.

54. The method of claim 52, wherein said anti-cancer compound is a histone deacetylase (HDAC) inhibitor.

55. The method of claim 54, wherein said HDAC inhibitor is a hydroxamic acid derivative, a Short Chain Fatty Acid (SCFA), a cyclic tetrapeptide, a benzamide derivative, or an electrophilic ketone derivative.

56. The method of claim 54, wherein said HDAC inhibitor is a hydroxamic acid derivative selected from the group consisting of SAHA, Pyroxamide, CBHA, Trichostatin A (TSA), Trichostatin C, Salicylbishydroxamic Acid, Azelaic Bishydroxamic Acid (ABHA), Azelaic-1-Hydroxamate-9-Anilide (AAHA), 6-(3-Chlorophenylureido) carpoic Hydroxamic Acid (3Cl-UCHA), Oxamflatin, A-161906, Scriptaid, PXD-101, LAQ-824, CHAP, MW2796, and MW2996.

57. The method of claim 54, wherein said HDAC inhibitor is a Cyclic Tetrapeptide selected from the group consisting of Trapoxin A, FR901228 (FK 228 or Depsipeptide), FR225497, Apicidin, CHAP, HC-Toxin, WF27082, and Chlamydocin.

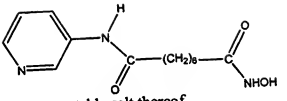
58. The method of claim 54, wherein said HDAC inhibitor is a Short Chain Fatty Acid (SCFA) selected from the group consisting of Sodium Butyrate, Isovalerate, Valerate, 4 Phenylbutyrate (4-PBA), Phenylbutyrate (PB), Propionate, Butyramide, Isobutyramide, Phenylacetate, 3-Bromopropionate, Tributyrin, Valproic Acid and Valproate.

59. The method of claim 54, wherein said HDAC inhibitor is a Benzamide derivative selected from the group consisting of CI-994, MS-27-275 (MS-275) and a 3'-amino derivative of MS-27-275.

60. The method according to claim 54, wherein said HDAC inhibitor is an electrophilic ketone derivative selected from the group consisting of a trifluoromethyl ketone and an α-keto amide.

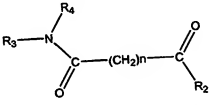
61. The method according to claim 54, wherein said HDAC inhibitor is a natural product, a psammaplin or Depudecin.

62. The method according to claim 54, wherein said HDAC inhibitor is pyroxamide, represented by the structure:



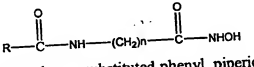
or a pharmaceutically acceptable salt thereof.

63. The method of claim 54, wherein said HDAC inhibitor is represented by the structure:



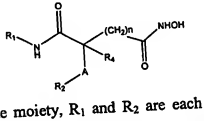
wherein R_3 and R_4 are independently a substituted or unsubstituted, branched or unbranched alkyl, alkenyl, cycloalkyl, aryl, alkyloxy, aryloxy, arylalkyloxy, or pyridine group, cycloalkyl, aryl, aryloxy, arylalkyloxy, or pyridine group, or R_3 and R_4 bond together to form a piperidine group; R_2 is a hydroxylamino group; and n is an integer from 5 to 8, wherein said HDAC inhibitor.

64. The method of claim 54, wherein said HDAC inhibitor is represented by the structure:



wherein R is a substituted or unsubstituted phenyl, piperidine, thiazole, 2-pyridine, 3- pyridine or 4-pyridine and n is an integer from 4 to 8.

65. The method of claim 54, wherein said HDAC inhibitor is represented by the structure:



wherein A is an amide moiety, R_1 and R_2 are each selected from substituted or unsubstituted aryl, arylalkyl, naphthyl, pyridineamino, 9-purine-6-amino, thiazoleamino, aryloxy, arylalkyloxy, pyridyl, quinolinyl or isoquinolinyl; R_4 is

hydrogen, a halogen, a phenyl or a cycloalkyl moiety and n is an integer from 3 to 10.

66. The method of claim 52, wherein the anti-cancer agent is an alkylating agent selected from the group consisting of bischloroethylamines, aziridines, alkyl alkone sulfonates, nitrosoureas, nonclassic alkylating agents and platinum compounds.

67. The method of claim 52, wherein the anti-cancer agent is an antibiotic agent selected from the group consisting of doxorubicin, daunorubicin, epirubicin, idarubicin and anthracenedione, mitomycin C, bleomycin, dactinomycin, and plicatomycin.

68. The method of claim 52, wherein the anti-cancer agent is an antimetabolic agent selected from the group consisting of floxuridine, fluorouracil, methotrexate, leucovorin, hydroxyurea, thioguanine, mercaptopurine, cytarabine, pentostatin, fludarabine phosphate, cladribine, asparaginase, and gemcitabine.

69. The method of claim 68, wherein said antimetabolic agent is gemcitabine.

70. The method of claim 52, wherein the anti-cancer agent is an hormonal agent selected from the group consisting of an estrogen, a progestogen, an antiesterogen, an androgen, an antiandrogen, an LHRH analogue, an aromatase inhibitor, diethylstibestrol, tamoxifen, toremifene, fluoxymesterol, raloxifene, bicalutamide, nilutamide, flutamide, aminoglutethimide, tetrazole, ketoconazole, goserelin acetate, leuprolide, megestrol acetate, and mifepristone.

71. The method of claim 52, wherein the anti-cancer agent is a plant-derived agent selected from the group consisting of vincristine, vinblastine, vindesine, vinzolidine, vinorelbine, etoposide teniposide, paclitaxel and docetaxel.

72. The method of claim 52, wherein the anti-cancer agent is a biologic agent is selected from the group consisting of immuno-modulating proteins, monoclonal antibodies against tumor antigens, tumor suppressor genes, and cancer vaccines.

73. The method of claim 21, wherein the immuno-modulating protein is selected from the group consisting of interleukin 2, interleukin 4, interleukin 12, interferon El interferon D, interferon alpha, erythropoietin, granulocyte-CSF, granulocyte, macrophage-CSF, bacillus Cahnette-Guerin, levamisole, and octreotide.

74. The method of claim 73, wherein the tumor suppressor gene is selected from the group consisting of DPC-4, NF-1, NF-2, RB, p53, WTl, BRCA, and BRCA2.

75. The method of claim 52, wherein the anti-cancer agent is a differentiation inducing agent.

76. The method of claim 52, wherein the therapeutic effect of SAHA and said anti-cancer agent is additive.

77. The method of claim 52, wherein SAHA sensitizes cancer cells in the patient to said anti-cancer agent.

78. The method of claim 52, wherein said anti-cancer agent sensitizes cancer cells in the patient to SAHA.

79. The method of claim 52, wherein SAHA and said anti-cancer agent are administered simultaneously.

80. The method of claim 52, wherein SAHA and said anti-cancer agent are administered sequentially.

81. The method of claim 80, wherein SAHA is administered prior to administering said anti-cancer agent.

82. The method of claim 80, wherein SAHA is administered after administering said anti-cancer agent.

83. The method of claim 52, wherein SAHA is administered orally, parenterally, intraperitoneally, intravenously, intraarterially, transdermally, sublingually, intramuscularly, rectally, transbuccally, intranasally, liposomally, via inhalation, vaginally, intraoccularly, via local delivery by catheter or stent, subcutaneously, intraadiposally, intraarticularly, intrathecally, or in a slow release dosage form.

84. The method of claim 52, wherein said anti-cancer agent is administered orally, parenterally, intraperitoneally, intravenously, intraarterially, transdermally, sublingually, intramuscularly, rectally, transbuccally, intranasally, liposomally, via inhalation, vaginally, intraoccularly, via local delivery by catheter or stent, subcutaneously, intraadiposally, intraarticularly, intrathecally, or in a slow release dosage form.

85. The method of claim 52, wherein SAHA is administered orally in a pharmaceutical composition comprising SAHA and a pharmaceutically acceptable carrier or diluent.

86. The method of claim 85, wherein said SAHA composition is contained within a gelatin capsule.

87. The method of claim 86, wherein said carrier or diluent is microcrystalline cellulose.

88. The method of claim 87, wherein said SAHA composition further comprises sodium croscarmellose as a disintegrating agent.

89. The method of claim 88, wherein said SAHA composition further comprises magnesium stearate as a lubricant.

90. The method of claim 52, wherein said SAHA composition is administered once-daily, twice-daily or three times-daily.

91. The method of claim 90, wherein said SAHA composition is administered once daily at a dose of about 200-600 mg.

92. The method of claim 90, wherein said SAHA composition is administered twice daily at a dose of about 200-400 mg.

93. The method of claim 90, wherein said SAHA composition is administered three times daily at a dose of about 200-400 mg.

94. The method of claim 90, wherein said SAHA composition is administered twice daily at a dose of about 200-400 mg intermittently.

95. The method of claim 94, wherein said SAHA composition is administered three to five days per week.

96. The method of claim 94, wherein said SAHA composition is administered three days a week.

97. The method of claim 96, wherein said SAHA composition is administered at a dose of about 200 mg.

98. The method of claim 96, wherein said SAHA composition is administered at a dose of about 300 mg.

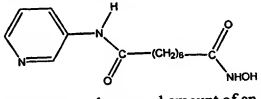
99. The method of claim 96, wherein said SAHA composition is administered at a dose of about 400 mg.

100. The method of claim 52, wherein the cancer is selected from the group consisting of a leukemia, a lymphoma, a myeloma, a sarcoma, a carcinoma, a solid tumor or any combination thereof.

101. The method of claim 52, wherein the cancer is selected from the group consisting of cutaneous T-cell lymphoma (CTCL), noncutaneous peripheral T-cell lymphoma,

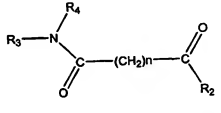
lymphoma associated with human T-cell lymphotrophic virus (HTLV), adult T-cell leukemia/lymphoma (ATLL), acute lymphocytic leukemia, acute nonlymphocytic leukemia, chronic lymphocytic leukemia, chronic myelogenous leukemia, Hodgkin's disease, non-Hodgkin's lymphoma, multiple myeloma, mesothelioma, childhood solid tumors such as brain neuroblastoma, retinoblastoma, Wilms' tumor, bone cancer and soft-tissue sarcomas, common solid tumors of adults such as head and neck cancers (e.g., oral, laryngeal and esophageal), genito urinary cancers (e.g., prostate, bladder, renal, uterine, ovarian, testicular, rectal and colon), lung cancer, breast cancer, pancreatic cancer, melanoma and other skin cancers, stomach cancer, brain cancer, liver cancer, adrenal cancer, kidney cancer, thyroid cancer, basal cell carcinoma, squamous cell carcinoma of both ulcerating and papillary type, metastatic skin carcinoma, medullary carcinoma, osteo sarcoma, Ewing's sarcoma, veticulum cell sarcoma, Kaposi's sarcoma, neuroblastoma and retinoblastoma.

102. A method of treating cancer in a subject in need thereof, comprising the step of administering to the subject a first amount of pyroxamide or a pharmaceutically acceptable salt or hydrate thereof, represented by the structure:



in a first treatment procedure, and a second amount of an anti-cancer agent in a second treatment procedure, wherein the first and second amounts together comprise a therapeutically effective amount.

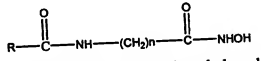
103. A method of treating cancer in a subject in need thereof, comprising the step of administering to the subject a first amount of a compound represented by the structure:



wherein R_3 and R_4 are independently a substituted or unsubstituted, branched or unbranched alkyl, alkenyl, cycloalkyl, aryl, alkyloxy, aryloxy,

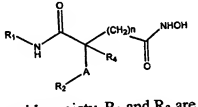
arylalkyloxy, or pyridine group, cycloalkyl, aryl, aryloxy, arylalkyloxy, or pyridine group, or R_3 and R_4 bond together to form a piperidine group; R_2 is a hydroxylamino group; and n is an integer from 5 to 8, or a pharmaceutically acceptable salt or hydrate thereof in a first treatment procedure, and a second amount of an anti-cancer agent in a second treatment procedure, wherein the first and second amounts together comprise a therapeutically effective amount.

104. A method of treating cancer in a subject in need thereof, comprising the step of administering to the subject a first amount of a compound represented by the structure:



wherein R is a substituted or unsubstituted phenyl, piperidine, thiazole, 2-pyridine, 3- pyridine or 4-pyridine and n is an integer from 4 to 8, or a pharmaceutically acceptable salt or hydrate thereof in a first treatment procedure, and a second amount of an anti-cancer agent in a second treatment procedure, wherein the first and second amounts together comprise a therapeutically effective amount.

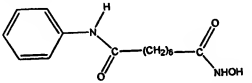
105. A method of treating cancer in a subject in need thereof, comprising the step of administering to the subject a first amount of a compound represented by the structure:



wherein A is an amide moiety, R_1 and R_2 are each selected from substituted or unsubstituted aryl, arylalkyl, naphthyl, pyridineamino, 9-purine-6-amino, thiazoleamino, aryloxy, arylalkyloxy, pyridyl, quinolinyl or isoquinolinyl; R_4 is hydrogen, a halogen, a phenyl or a cycloalkyl moiety and n is an integer from 3 to

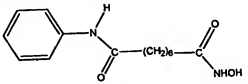
10, or a pharmaceutically acceptable salt or hydrate thereof in a first treatment procedure, and a second amount of an anti-cancer agent in a second treatment procedure, wherein the first and second amounts together comprise a therapeutically effective amount.

106. A method of selectively inducing terminal differentiation of neoplastic cells in a subject and thereby inhibiting proliferation of said cells in said subject, said method comprising the step of administering to said subject a first amount of suberoylanilide hydroxamic acid (SAHA) or a pharmaceutically acceptable salt or hydrate thereof, represented by the structure:



in a first treatment procedure, and a second amount of an anti-cancer agent in a second treatment procedure, wherein the first and second amounts together comprise an amount effective to induce terminal differentiation of said cells.

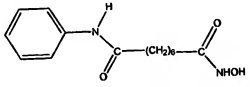
107. A method of selectively inducing cell growth arrest of neoplastic cells in a subject and thereby inhibiting proliferation of said cells in said subject, said method comprising the step of administering to said subject a first amount of suberoylanilide hydroxamic acid (SAHA) or a pharmaceutically acceptable salt or hydrate thereof, represented by the structure:



in a first treatment procedure, and a second amount of an anti-cancer agent in a second treatment procedure, wherein the first and second amounts together comprise an amount effective to induce cell growth arrest of said cells.

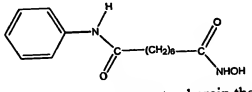
108. A method of selectively inducing apoptosis of neoplastic cells in a subject and thereby inhibiting proliferation of said cells in said subject, said method comprising the step of administering to said subject a first amount of suberoylanilide

hydroxamic acid (SAHA) or a pharmaceutically acceptable salt or hydrate thereof, represented by the structure:



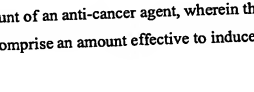
in a first treatment procedure, and a second amount of an anti-cancer agent in a second treatment procedure, wherein the first and second amounts together comprise an amount effective to induce apoptosis of said cells.

109. An in-vitro method of selectively inducing terminal differentiation of neoplastic cells and thereby inhibiting proliferation of said cells, said method comprising the step of contacting the cells with a first amount of suberoylanilide hydroxamic acid (SAHA) or a pharmaceutically acceptable salt or hydrate thereof, represented by the structure:



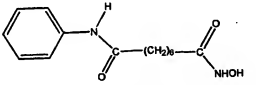
and a second amount of an anti-cancer agent, wherein the first and second amounts together comprise an amount effective to induce terminal differentiation of said cells.

110. An in-vitro method of selectively inducing cell growth arrest of neoplastic cells and thereby inhibiting proliferation of said cells, said method comprising the step of contacting the cells with a first amount of suberoylanilide hydroxamic acid (SAHA) or a pharmaceutically acceptable salt or hydrate thereof, represented by the structure:



, and a second amount of an anti-cancer agent, wherein the first and second amounts together comprise an amount effective to induce cell growth arrest of said cells.

111. An in-vitro method of selectively inducing apoptosis of neoplastic cells and thereby inhibiting proliferation of said cells, said method comprising the step of contacting the cells with a first amount of suberoylanilide hydroxamic acid (SAHA) or a pharmaceutically acceptable salt or hydrate thereof, represented by the structure:



and a second amount of an anti-cancer agent, wherein the first and second amounts together comprise an amount effective to induce apoptosis of said cells.